

"Note on the Effects produced on Rats by the Trypanosomata of Gambia Fever and Sleeping Sickness." By H. G. PLIMMER, F.L.S. Communicated by Dr. C. J. MARTIN, F.R.S. Received December 1, 1904,—Read January 19, 1905.

(From the Laboratories of the Lister Institute of Preventive Medicine.)

The following observations are of interest in connection with the view that Gambia Fever may be the early stage of the disease which ends as Sleeping Sickness, which is held by some: and with the question of the causal relationship of the Trypanosoma found in Sleeping Sickness to the disease.

The Trypanosomata used in the experiments mentioned below were given to me by Col. Bruce, F.R.S.; that of Gambia Fever having been taken from a monkey inoculated in Africa from a native boy suffering from Gambia Fever, and that of Sleeping Sickness from a monkey inoculated in Africa from a case of this disease.

Gambia Fever.

Fourteen rats were inoculated with the Trypanosoma of Gambia Fever at intervals between December 11, 1903, when I received the first rat from Col. Bruce, and August 24, 1904, all of which are dead. In each case the Trypanosomata were present in the blood, the first appearance of them there being about four weeks after inoculation: and towards the end of the disease they were present in large numbers. The average time between inoculation and death was two months and twelve days. In each case the spleen was enormously enlarged; there was considerable hyperplasia of the follicles, and the pulp was congested in varying degrees. There was also marked hyperplasia of the lymphatic glands, especially of those of the mesenteric and mediastinal areas. There was congestion of the liver, with cloudy swelling of its epithelium, and the same condition in a lesser degree was found in the kidney. In no case was there any paralysis, or any nervous symptoms, except that just before death the animals became heavy and apathetic. *Post mortem* the capillaries of the brain contained large numbers of the organisms.

Sleeping Sickness.

Three rats were inoculated on December 1, 1903, from a monkey which had been brought home by Col. Bruce from Africa after inoculation with cerebro-spinal fluid from a case of Sleeping Sickness. At no time were any Trypanosomata found in the blood of these rats, even after it had been centrifugalised *post mortem*. One rat became

paralysed in both hind legs on May 12, 1904, and died on May 23 ; the second became paralysed similarly on August 2, and died on August 30 ; and the third became paralysed on August 28, and died on September 8. On *post mortem* examination no naked-eye lesions of any organ were found, and the spleens were not enlarged. The blood was citrated and centrifugalised, and the organs were mashed and washed with normal salt solution and centrifugalised, but in no case were any *Trypanosomata* found. Portions of the extracts of liver, spleen, and spinal cord were injected into other rats, but up to the present these show no sign of illness. In the mashed spinal cord of each of these rats the characteristic *Trypanosomata* were found in small numbers, but none were found in the brains which were examined in the same way. The three rats were of the same kind, black and white ; and the youngest of the three died first and the eldest last.

(1) These experiments go to show that the two diseases—*Gambia Fever* and *Sleeping Sickness*—from which the organisms were obtained, are distinct ; the duration of the diseases, the symptoms, and the *post mortem* appearances being quite different.

It is evident that these two organisms are quite separate and distinct, as their different effects on similar animals indicates ; moreover, they are also morphologically distinct when grown in similar animals, that of *Gambia Fever* being longer, generally larger, and more easily stainable than the stumpy, large-vacuolated, badly staining *Trypanosoma* of *Sleeping Sickness*.

The fact of the clinical observation that *Gambia Fever* not infrequently appears to terminate with all the symptoms of *Sleeping Sickness* may quite possibly be explained by a double infection. For, in both rats and monkeys the one *Trypanosoma* does not interfere with the other, but the more active organism—that of *Gambia Fever* in the cases of rats and monkeys—kills in about the same time, whether inoculated before, with, or after that of *Sleeping Sickness*.

(2) There can be no question, from the above experiments, of the susceptibility of the rat to the *Trypanosoma* of *Sleeping Sickness*.

(3) These experiments show that the inoculation of the *Trypanosoma* of *Sleeping Sickness* into rats gives rise to no obvious symptoms for many months, nor are *Trypanosomata* discoverable in the blood by microscopic examination. But, after a period of from six to nine months paraplegia occurs, leading to the death of the animal ; and *post mortem* the organisms are found only in the spinal cord. The organisms are thus in rats, as sometimes in man, entirely confined to the nervous system ; whereas in monkeys they are, in my experience, always generalised at some period of the disease.

Addendum, January 19, 1905.—The three rats inoculated on August 30, 1904, with extract of spinal cord have become paraplegic: one on December 14, 1904, one on January 2, 1905, and the third on January 11, 1905. No Trypanosomata have been seen in the blood of these animals, and, apart from paralysis of the hind limbs, they show no signs of ill health.

The spinal cord shows amoeboid forms of the Trypanosomata in its tissue, and a considerable cellular exudation around the vessels; this lesion Dr. Mott found to be characteristic of cases of human Sleeping Sickness, and it is not found in monkeys, which have been the animals principally used in experimental work on this disease.

“Further Histological Studies on the Localisation of Cerebral Function.—The Brains of *Felis*, *Canis*, and *Sus* compared with that of *Homo*.”* By ALFRED WALTER CAMPBELL, M.D. Communicated by Professor SHERRINGTON, F.R.S. Received December 3, 1904,—Read January 19, 1905.

(Abstract.)

The present study is founded on an exhaustive examination of the cerebral cortex of *Felis domesticus*, *Canis familiaris*, and *Sus communis*, conducted on lines similar to those followed in the original work.

A recurrence in the lower mammal of distinctive types of cortex, akin to those recognised in the human brain, has allowed a subdivision of the surface into the following areas:—

Crucial or motor; posterocrucial or sensory; parietal; visual; ectosylvian; limbic; rhinic; extrarhincic; frontal.

By a study of the distribution of these areas many functional analogies and structural homologies, previously unknown or misinterpreted, are made clear.

Crucial or Motor Area.—Giant cells of Betz characterise the motor cortex, but these elements appear not to be so highly specialised in *Sus* as in *Felis* and *Canis*. Such cells reside in what we may call the cruciate zone, and it is maintained that this field is functionally and morphologically akin to the motor area, as defined by Professors Sherrington and Grünbaum in the anthropoid ape, and by myself in man. It is held that a small indentation, called the “compensatory ansate” sulcus, and the sulcus coronalis are respectively interchangeable with the upper and lower constituents of the primate fissure of Rolando, chiefly because they, like the fissure of Rolando, limit the

* This paper is an addendum to the work presented to the Royal Society in November, 1903 (see ‘Proceedings,’ vol. 72, p. 488). The complete work will be published shortly in full by the Cambridge University Press, by aid of a subsidy from the Royal Society.